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Volkmann's ischemic contracture

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VOLKMANN'S ISCHEMIC CONTRACTURE

SENIOR THESIS

Frank J. Bertoneclj

1935

UNIVERSITY OF NEBRASKA COLLEGE OF MEDICINE

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PREFACE

In undertaking the writing of this thesis I am fully aware of the difficulty in maintaining the standard set by the medical profession in this respect. It has been my aim to incorporate the more recent facts and points of view on the subject of Volkmann's ischemic contracture without sacrificing the interesting history and evolution of the subject and in a small way to perpetuate the memory of some of our indomitable men of medicine.

As Holt states " In an age where deference to authority and adherence to tradition have been rapidly giving way to an attitude of scientific inquiry" I may be forgiven in presenting this subject in a less didactic manner; And while I have not hesitated in presenting the opinions of several writers I have attempted to present a quasicomplete evidence and theory upon which they are based and backing it up with a verified comprehensive bibliography in order to limit the length of the manuscript as far as possible and thus retain its practicality.

The opinions and conclusions expressed in these pages are wholly others and I have no apologies

to offer in view of my embryonal experience, however, the inquisition is spontaneous and original and this source has been a great pleasure since it has explained to my full satisfaction, the question uppermost in my mind since I first heard that impressionable word main-en-griffe in my first year in medical school. There has been very little said about the condition in classes, clinics and text books except to make mention of it.

I have attempted a rather complete bibliography in view of the fact that the treatment of the condition has been omitted on the advice of Dean Poynter because this phase is subject to too rapid mutability. The bibliography is sufficiently complete so that one can refer to it for the necessary information on this phase of the condition.

I have attempted to deal more with the interpretation and pathogenesis of the condition without making it too voluminous to be practical and in this I was not a little influenced by the advice of Holt who says "Intelligent treatment, whether it be rational or empirical, depends upon the thorough understanding of the nature of disease."

The general practitioner will find here a convenient summary of the clinical and biological aspects of Volkmann's ischemic contracture which have not been treated in this manner since Brooks reviewed the subject in 1922; there have appeared since then some important findings on the condition but which are scattered among numerous publications.

Many articles have been written on this subject and still all interested parties agree that not sufficient emphasis is placed upon it nor can a subject of such moment be too often repeated.

VOLKMANN'S ISCHEMIC CONTRACTURE

Synonyms - This condition has been variously called Volkmann's contracture, after Richard Volkmann, a german surgeon, (1830)-1889, who was generally accredited with having first described it, Volkmann-Leser paralysis, Volkmann's ischemic paralysis, Ischemic myositis, Ischemic contracture, Contracture myositis and Contracture.

These variable appellations are the result of differences of opinion as regards the etiological factor of the condition and who should be thus honored with having first recognized it.

Definition - Meyerding suggested the following definition Volkmann's ischemic contracture is a deformity, usually of fore-arm, wrist and hand, with or without paralysis; complicating trauma, usually fracture, and producing varying degrees of disability.

Dickson brought out the fact that Volkmann's contracture is a condition which causes severe deformity and complete or almost complete loss of function.

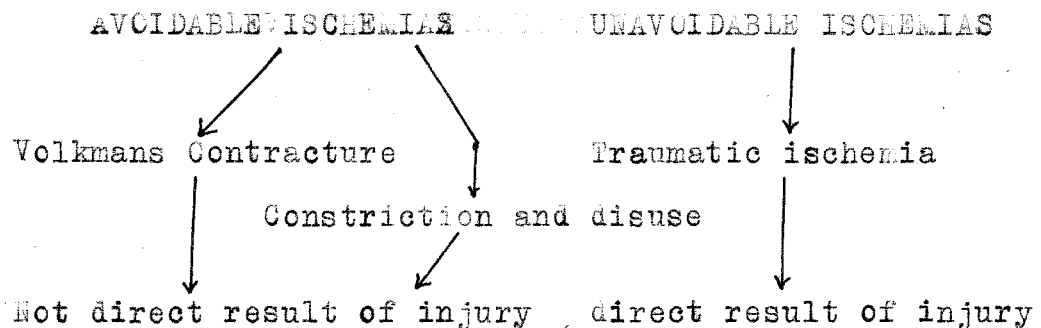
Lewis said "Ischemic myositis is one of the most disabling lesions associated with fractures."

Volkmann's contracture is a condition which is of particular interest to anyone who handles fractures first, because of the vicious complications that one

may have following fractures, or injuries, near the elbo joint or fore-arm, and again because of different opinions advanced by the authorities, both as to pathology and etiology, particularly the latter.

Volkmann's contracture is extremely important because it is a constant potential complication of every extensive, deep or severe injury to the knee and elbo and their immediate proximity.

Girdlestone classified ischemias into the following classes in order that there would be less chance of confusing Volkmann's ischemic contracture with some other conditions which are apt to simulate it very closely in their earlier manifestations but the outcome of which are very much less vicious and disconcerting.



History - There seems to be some slight disagreement as to the exact time Volkmann described and announced the condition which bears his name.

Meyerding beleives that Volkmann was the first to describe the condition in 1869. Lewis, however, maintains that Volkmann, in 1880, described this condition of degeneration, fibrosis, and contraction of the tissues of the fore-arm, affecting principally the muscles, as being produced by prolonged ischemia.

Jones found in the literature that Volkmann in 1875 described an ischemic contracture of the hand which he ascribed to tight bandaging and believed the deformity was due to contracture myositis and not to primary nerve pressure palsy. Jones puts the date of the first classical article as being published in 1881. He said, "The contributions of Volkmann and Leser are very complete, and have required but little revision or addition for 50 years.

Thomas accepts Volkmanns general clinical description as being substantially accurate and hardly to be bettered. except on certain points, where a study of the cases obviates error, his views must be changed. or at least modified.

Jepson disagrees with the whole lot and maintains that the credit of calling attention to the condition and establishing it as a real entity belongs to Leser. He found that Leser in 1884 reported seven cases and that he believed the condition was caused by deprivation of muscles of oxygen. Leser, however, failed to give any theory as to how this lack of oxygen was caused.

Lewis thinks that Bardenhuer made the greatest contribution to the subject of ischemic contracture.

The first american writer to describe the microscopical changes in the muscle as a result of the ischemia is Barneys, according to Thomas.

Clinical course - The symptom-complex is not constant but there is some variation in each individual case particularly as regards the degree or severity of signs and symptoms. The resulting condition of the muscle is always the same.

When the contracture is impending the patient usually has fever caused probably by the absorption of degenerated muscle tissue. There might be a decided general reaction as is observed by several writers.

The following case, reported by Nelson, I find, illustrates most typically the clinical course of the condition.

Girl, age 9 years, fell on her left hand fracturing the lower end of the humerus with posterior displacement of the distal fragment. Arm strapped at right angles.

--After two hours fracture reduced. Elbo felt brawny. No crepitus suggesting interposition of soft tissue between fracture fragments. No radial pulse? Arm at right angle.

--After six hours - Arm very painful - induration increased in severity and extent. Strapping removed.

--After eight hours - Induration so severe that pres-

sure had no effect. Pain increased.

--After ten hours - pain the same - appearance that of impending gangrene - limb cold - blebs formed on skin - skin dusky bluish.

--After eleven hours - four inch incision made at area of greatest induration lateral to the antecubital fossa - Findings, divided small artery, median nerve and brachial artery interposed in fracture site.

--Subsequent progress - complete recovery in six months - slight temperature curve during first week and in absence of infection attributed to absorption of the hematoma.

The skin following the swelling becomes thin and delicate and deprived of subcutaneous fat.

The length of time that the condition exists which results in contracture before the contracture actually occurs is variable according to Meyerding and others.

Taylor took particular cognizance of the constitutional reactions associated with this condition and found even more varied systemic symptoms as indicated by a temperature of from 102 to 104 degrees fahrenheit, headache, malaise, anorexia, etc.

Within two weeks the swelling subsides and antecubital swelling and induration disappears.

Occurrence - The condition is not limited to the arm and leg but can occur any where in the body although it is rare else where as conditions for complete ischemia are not favorable in other locations as has been demonstrated experimentally by Cravener and others.

Meyerding in a statistical analysis found that in more than ninety percent of cases the upper extremity is involved. The condition is apt to occur at any age, however, the consensus of opinion seems to be that the condition occurs most frequently by far at ages from eight to eighteen. Cases have actually been reported of its occurring at ages from three to sixty one, and in each a typical deformity resulting.

The condition probably is not as rare as it would appear to be from the scarcity of reported cases but Meyerding believes that physicians hesitate to report results, in cases in which lack of care or unskilled care present possible grounds for malpractice suits.

It is interesting to note that though ninety percent of Volkmann's contractures occur in the fore-arm, the first reported case of the condition, by Volkmann, should be that of one occurring in the leg.

There is a case, reported by Stone , where the only muscle involved was the pronator quadratus and this limitation of the condition to a muscle or even a part of one muscle, was observed by Jones earlier. In that particular case only a small portion of the muscle was involved.

Murphy in pointing out the universal occurrence of the contracture, as the biceps for instance, writes that he has seen upwards of fifty cases in five years of this type of condition involving the upper arm.

Jepson found that the extensor muscles are usually not involved in myositic change although tension on the extensors musculature is unavoidable due to hyperflexion of metacarpophalangeal joints, however, Murphy reports two cases that were definitely caused by splints, of volar extensors.

In the forty years following Volkmann's original report, Carlson was able to find only 103 reported cases on record. In the Mayo Clinic there

were 128 cases seen there from 1910 - 1927.

The condition is thought to be quite frequent by Thomas but he expressed surprise to find that the condition was not more frequently reported although he avers that it is probably due to the fact that Volkmann's contracture is thought to be due to too tight bandaging or neglect, which is not true as we shall see.

Thomas observed that the condition occurred more often in males than in females and attributed this to the greater liability of the male to accidents. He also remarked that the predominance of Volkmann's contracture in children is very striking.

In this connection Stone observed a distressing number of Volkmann's contractures in the childrens hospitals. He said, "Children have trouble more frequently than adults because, I believe adults do not submit to the pain that the children are compelled to bear, adults rip off too tight bandages."

Although Stone seems to be of the opinion that tight bandaging is the only etiological factor in the production of the condition, Lewis finds that

at least 8 percent of cases of ischemic paralysis occur without a cast or constricting bandage having been applied.

Volkman's contractures are observed most frequently after supracondylar fractures and fractures of the lower end of the humerus according to Lewis.

Cravener says, "Certain entities in medicine have become inseparable ---- similarly, the condition of Volkman's contracture the reaction word to which is fore-arm!"

Etiology - Meyerdig is of the opinion that the etiology of Volkmann's contracture is still very far from being worked out satisfactorily.

Beck believed that Volkmann's theory of pure myogenic origin of ischemic muscle contracture seems correct. This does not mean that the muscle really dies, as in that case gangrene would set in. A primary nerve effect is not present. Sensory and trophic phenomena complicate the picture but have no etiologic relationship. The latter may be due to associated nerve lesions from either the original trauma or secondary compromising of the nerves in the scarred muscle. There is no substantiating proof that the lesion is due to the alterations in the sympathetic fibers.

Murphy in reviewing the work of Bardenheuer found that he was of the opinion that the cells are poisoned by the metabolic products as a result of venous stasis. Murphy himself believes that it is the pressure on the muscle which causes destruction of the cells. He says, "Certain it is that severe contusion of the muscle of an extremity, with or without fracture or external wound or evidence of injury, may be followed by this contracture"

The obstruction is in the veins and not in the arteries, as the great edema always indicated obstruction to return circulation and not to arterial circulation.

Brooks is of the opinion that simple hemorrhage in the muscle with or without interference with the arterial nerve supply of the muscle does not cause hemorrhage, edema and degeneration of the muscle fibers and an acute inflammatory process which progressed to the more or less fibrosis of the muscle. The slowing of circulation is the first phenomena observed in the process of inflammation. In blocking the veins the circulation is thus primarily slowed down and all of the succeeding manifestations of inflammation and repair follow.

Meyerding reasons that since it is impossible to have anemia and inflammation at the same time it stands to reason that it is hardly likely that since we do have inflammatory reactions in impending Volkmann's contracture it is not complete ischemia, however we do know that the muscle atrophy does not manifest itself until inflammation has acted and until it begins to subside, so we may assume that inflammation is the primary condition and occurring in the regions of the elbow and knee where the muscular sheaths are of a firm, all enclosed nature,

it produces such a degree of ischemia that the inflammation is itself limited and conditions are so rapidly produced which are compatible with inflammatory reactions.

Jones showed that that obstruction of artery produces only temporary ischemic contracture. Obstruction of vein causes the severest kind of ischemic contracture providing that obstruction is sudden and complete. It is his opinion that deformity does not occur if obstruction is gradual.

Nelson is of the opinion that it is a condition of muscle tissue anoxemia which is responsible and which must be rapid. He seems to have quite conclusive proof as illustrated by one of his cases in which there was a sudden almost complete obstruction of arterial blood supply and fairly rapid venous obstruction due to hematoma as a result of tearing of a lesser colateral artery.

Meyerding contends that the condition cannot be caused by circulatory disturbance alone since occlusion of arterial vessels causes dry gangrene with flaccid paralysis and venous obstruction results in wet gangrene, this led Thomas to believe

that nerve involvement alone was responsible, but here again it has been demonstrated that nerve destruction results in flaccid paralysis and elongation of the muscles which still retain their elasticity though very atrophic; while on the other hand, as in Volkmann's contracture, the muscle cells themselves are more or less destroyed and fibrosed and degeneration results in a shortened, atrophied, hard, inelastic, cord like scar tissue which cannot function like normal tissue.

Brooks found in those instances in which a bandage or splint is applied to an extremity presumably never so tight as to obstruct immediately or completely the circulation distal to the constriction and in which follows in the course of a few hours great pain and cyanosis which make the condition no longer bearable, and in which the removal of the splint is followed by swelling, heat, tenderness, and rapidly developing contracture, one is forced to the assumption that the etiological factor is either venous obstruction of an acute type or a temporary pressure anemia followed by a reestablishment of circulation through the damaged tissue. The ease with which the

former is reproduced experimentally is evidence of its being the most important etiological factor, by constricting dressings in the treatment of fractures.

That the classical picture of Volkmann's ischemic contracture could only be explained on the basis of acute venous obstruction would seem quite reasonable. As a matter of fact, however, it is customary to refer to every case of deformity in this region following the application of splints or plaster casts, tight bandaging, tourniquet, as Volkmann's contracture. Undoubtedly, some of these cases are due to pure pressure necrosis, entirely analogous to the decubitus ulcer, or pressure sore on heel, or over the anterior superior iliac spine. from continual pressure of a plaster cast.

The intrinsic pressure produced by the hematoma is stressed as the main factor by Marble. It has been demonstrated that this alone is sufficient to produce the condition and having this factor present, anything that will cause additional pressure is merely adding insult to injury and increasing the possibility of a complication.

Lewis further emphasizes this, the importance of a subfascial hematoma, in the production of ischemic contracture and stresses the fact that it has not been fully appreciated.

Volkman, quoted by Jones, demonstrated that similar contractures have resulted following blood stasis due to compression with the esmark bandage, also to injuries of the large vessels and from exposure to cold.

Substantiating this idea was Page, quoted by Jones, in an able communication affirmed that this interesting contracture was due to combined influences of pressure, fixation and ischemia.

As proof that cold can produce this condition, Kraske, quoted by page and reviewed by Jones, describes the microscopical examination of the muscle in the leg amputated for gangrene due to exposure, showed that the appearance of the muscle tissue corresponded closely to the muscle changes in a limb which had for a time been rendered bloodless by circular constriction. The ischemia induced in one case by lowered temperature and in the other by circular compression produced a necrobiosis of the contractile substance to which was added some precedent inflammation in the perymesion internum and

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regenerative processes in the surviving muscle fibers.

Murphy believes that eighty percent of these contractures follow fractures, especially fractures in the neighborhood of the elbow joint, and among these the supracondylar fracture stands out prominently.

Carlson maintains that it occurs where there is no displacement of fracture fragments, regardless of where the fracture is located, and even in injuries not associated with fracture.

Jones further emphasizes the fact that not all contractures are due to fractures and dislocations but may be due to other injuries.

However this may be Eikenberry showed that out of twenty one cases of Volkmann's ischemic contractures he saw in three years, every one of them had, without a single exception, a persistent posterior displacement of the distal fragment of the humerus in fractures of the supracondylar type.

In regards to tight bandaging as an etiological factor, Thomas writes that this is by no means true, though some type of interference with the circulation is probably necessary for the production of Volkmann's contracture.

Littlewood, quoted by Jones, basing his conclusion on two cases, neither had splint sores, denies the casual relationship of splints to deformity.

Nelson further substantiated the opinion that tight bandaging is not necessary for the production of the contracture.

The condition is so often alluded to as a neuro-muscular phenomena and there has been considerable argument put forth that the condition is caused primarily as a result of nerve involvement in the injury, this is not surprising considering that the nerve is seldom spared either at the time of the injury or the contracture or when we consider the probable etiology.

Jones dismisses this idea with the following explanation "Ischemic contracture is primarily a muscle lesion as some would contend that it is of neurogenic nature, and this opinion is quite universally accepted.

Bradley, quoted by Jepson, recently contributed the thought of atrophic changes being brought about by chemical liquefaction of the tissue

proteins acted upon by enzymes. Peptoids and amino acids are formed as end products. In some cases of atrophy, digestion of the tissue proteins is further facilitated by phagocytosis, but this, of course, is a secondary process. He believed that this increase of connective tissue and of adipose tissue was a compensatory invasive process from the intermuscular septum into the atrophied muscle and a phenomenon secondary to the atrophy of muscle itself.

Thomas mentions the importance of keeping the factor of embolism in mind as an etiological possibility in the production of Volkmann's contracture since contractures have been reported as having followed emboli by Langer and Schloffer.

Lord reported a case of a hemophylia who had extensive subcutaneous hemorrhages in both fore-arms and subsequent development of Volkmann's contractures. This differed slightly from the conventional deformity in that the wrists were not flexed but he ascribes this to the fact that the two sets of muscles, flexors and extensors, were involved equally. He says "I have presented this case because of its rarity, having found no record of a case with

similar causation, and believe that ischemic myositis and neuritis - Volkmann paralysis can be produced by impairment of the circulation when neither splints nor tight bandages have been used.

Jepson is of the opinion that the almost universal involvement of the flexors but not of the extensors may be explained on anatomic grounds. On the anterior surface of the fore-arm the soft tissues, composed of flexor muscles, arteries, and nerves, overlies the radius and ulna, forming a good sized pad. Besides this, the body of the anterior flexors comes in direct contact with the source of injury, whether it be splints, bandages, casts or direct contact with the source of injury. The ulna is situated between the splint and the extensor muscles.

Lewis further supports this idea of anatomic relations in this region. He is quite convinced that the anatomic relations favor the production of ischemic contractures. The antecubital fossa is covered by dense fascia, as is also the popliteal fossa. Related to each fossa are large venous trunks which can easily be compressed by a hematoma developing beneath the fascia.

Gravener is of the opinion that the deep flexors of the deep flexor group are always most to be effected as is illustrated in almost every case of true Volk-mann's contracture by the flexion of the distal phalanges. This fact would lead one to conclude that since this is true and since the long flexors of the thumb are also involved that mechanics have a great deal to do with the production of the condition, this distinct and uniform deformity, that is the unrelieved congestion, which is greatest here due to their being the most bounded by fascial capsules. In attempting to explain why the condition occurs more often in the arm than leg, he states that "perhaps lack of specialization of the leg muscles masks the result of muscle damage".

It was expressed by Carlson that the etiology presents but one factor that all writers agree upon, and that is that the interference of the circulation is caused by pressure, but whether it is intrinsic or extrinsic or a combination of both, is the topic upon which opinions differ. intrinsic pressure is produced by hematoma and displaced fracture fragments. Extrinsic pressure is from tight bandaging, splints, casts, or forced position.

Brooks opines that the reproduction of areas of degeneration and subsequent fibrosis of muscles by temporary anemias is so difficult experimentally that it is rather unlikely that the exact set of conditions for its causation would be present in any one clinical instance.

Jepsons experiments did show that a combination of factors is concerned in the etiology of this contracture as nearly as he could determine.

Pathology - Jepson describes the following pathological changes on gross inspection..The muscular tissue is pale yellow, hard and board like, and the tendons are usually matted together.

Dickson further describes the gross changes. In the flexor group of muscles there are more or less wide spread areas of necrosis of muscle fibers and their replacement by dense connective tissue so that the bodies of the muscles contain few contractile fibers, are pale in color, hard and firm in texture and are bound together in a firm mass by adhesions.

Jones gives an account of the microscopical changes which is most complete. In many portions there is absence of sarcolemma nuclei, and but few individual muscle fibers. There is round cell infiltration, showing presence of inflammatory changes. Transverse striations are usually absent or at least very scarce. The whole process seems to be one of absorption and replacement of dead muscular tissue by fibrosis. The muscle acting as a non-septic, non-irritating foreign body, and being treated as such by the surrounding tissue.

Diagnosis - The earlier the diagnosis, and especially the recognition of the impending signs of the condition, the better the prognosis and this is probably more true of Volkmann's contracture than any other clinical condition. If the condition is diagnosed sufficiently early prognosis is very good and if treated at this time recovery is complete.

When the patient complains of severe pain, swelling, numbness and a change in color of the limb following injury to the limbs or following acute flexion in attempts at reduction it is extremely important to suspect impending contracture from standpoint of a good prognosis, and this stage is the crucial moment to keep in mind if one is going to prevent the deformity.

The condition is extremely easy to diagnose after the contracture has developed for when the hand is grasped a typical, claw-like, cold lifeless feeling is imparted, and the cord-like tendons stand out on the flexor side that are easily palpated.

Meyerding described the deformity as consisting of an arm with the elbow partially flexed, frequently there is evidence of sloughing, usually

along the flexors, near the elbow, and of atrophy of the muscles, characterized by shrinking. The pronator radii teres is frequently involved, and, because of shortening, fibrosis, prevents supination. The wrist is flexed while the metacarpophalangeal joints may be hyperextended and the terminal phalangeal joints contracted sharply.

The myositis with infiltration has usually developed before the characteristic swelling and position of the fingers has appeared.

It may be quite difficult at times to decide when the more acute signs and symptoms are those of impending contracture and when they are due to the trauma from the injury, however, if the symptoms of acute pain etc/ seem to be increasing it is better to err on the safe side.

In the early stages of impending contracture the skin over the antecubital fossa is apt to be ecchymotic. Usually tense and shiny.

Severe spontaneous pain extending over the entire forearm in cases of supracondylar fracture is a symptom of importance and should demand immediate attention.

Pain is increased by pressure, and not infrequently an infiltration is noticed in front of the wrist.

The spontaneous pain is usually severe and the patients complain bitterly. The pain is felt particularly in the antecubital fossa and along the course of the veins of the fore-arm. It is felt to some extent through out the arm.

Cravener warns that it is possible too that some of the delayed functional results of fractures of the leg are due, in part, to unrecognized fibrosis of the calf group.

Murphy says "The deformity is in itself diagnostic. It is the typical claw-finger that is intractable to subjective attempts to straighten it out " and this deformity he speaks of subsequently as the *Main-en-griffe*.

As observed by Murphy the sequence of events is as follows: The muscles become flaccid and powerless. Muscular rigidity is then noted and the painful contracture begins. This disappears in about sixty five to seventy hours, leaving the muscles again flaccid and very tender to pressure. After a few more days the swelling disappears and muscular

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tissue begins to be replaced by fibrous tissue.
muscles then become hard and resistant, and
muscular atrophy and fibrosis appears.

Differential diagnosis - I have given below a
classification of the possible changes in Volkmann's
contracture as worked out by Thomas, so that there
would be less chance of confusing this condition with
some other or thinking that it is free of compli-
cations of its own. I have found in the literature
that some investigators even thought they had worked
out a new etiological factor of the contracture by
misinterpreting some complication as constituting
the end result.

Classification

- (1) Motion defect - elbow joint - displaced bones
or projected fragments.
- (2) Limitation of supination and pronation.
- (3) Coldness or blueness of hand - circulatory
embarrassment.
- (4) Trophic changes - glossy skin - ulcers - blebs.
- (5) Nerve function disturbances - sensory ^{median} ulnar
radial
- (6) Atrophy or paralysis of the small muscles of the
hand supplied by these nerves.

Steindler gives a very able review of this phase of Volkmann's contracture. Changes in this condition may be classed as interstitial myositis with secondary changes degenerative in nature of the muscle fibers, very similar to what we find in other forms of myositis.

In infantile paralysis and in peripheral nerve lesions the ensuing paralytic changes in the muscle seem to be more those of parenchymatous degeneration with secondary infiltration.

Considering the ischemic contractures as primarily of interstitial myositis, one can more readily understand why this condition of contracture shows the gradual and almost irresistible tendency to contracture, as Volkmann noted, by tremendous resistance to all attempts at reduction from the very moment of its inception.

Complications - Meyerding believes that in Volkmann's ischemic contracture the peripheral nerves are usually involved secondarily, or they may be cut through, lacerated or otherwise injured at the time of the accident or during manipulation as in reduction of a fracture or the transportation of the patient to the hospital.

He also points out that the joints are not involved until the later stages when, atrophy of bone and contracture of the capsule are not uncommon.

Jepson is of the opinion that as a rule there is no change in the joints other than a tendency toward subluxation. The atrophy of joint capsule at the elbow or the wrist is attendant upon long standing contracture.

The possible complications as worked out by Thomas have been given above in the Differential diagnosis.

Prognosis - All the damage that is done as far as the myositis is concerned is done in the first seventy five hours after the injury, but the contraction of the infiltrated or replacement connective tissue and the flexion deformity of the fingers does not show itself fully until weeks have elapsed, from four to six, the fixation becoming permanent in from ten to twelve weeks. It then remains stationary, except for additional deformity which is produced by the growth of the length of the long bones, without a growth of muscle to correspond. This makes it appear as though the muscle were continuing to contract, which is not the case according to Murphy.

The latest and most complete prognostic outline is given by Meyerding who makes the following conclusion:

Group 1 - Deformity days or weeks duration - typical

typical claw-like hand. - arterial pulse good -

warm hand - all respond to Jones splint treatment.

Group 2 - Months duration - cold hands - diminished

pulse - fingers can still be brought out straight

wrists slightly flexed on bringing hands out straight

straight - less favorable response to Jones treatment.

hesitate to operate until fair trial.

Group 3 - Older patients - duration of the contracture

longer - only help surgical - tenotomy - neurolysis.

Group 4 No longer functional restoration and may have to amputate.

From a good prognostic standpoint the condition should not have been allowed to have gone beyond the stage of pain, of a severe nature, swelling, numbness, and change in color of the limb.

As has been said before and this cannot be stressed too much, the earlier the diagnosis and especially the recognition of its premonitory signs the better the prognosis. If the condition is recognized as that of impending contracture and proper treatment instituted recovery is complete.

It has been pointed out that Volkmann's contracture may be so severe and complete that amputation is necessary.

The prognosis varies with the completeness of the destruction of the muscle tissue as was shown by Thomas in a review of cases reported in the literature.

Prophylaxis - Driver stresses the fact that there is perhaps nothing in the realm of medicine and surgery in which prevention is as imperative,,and is as easily attained, as in impending volkmann's ischemia.

Likewise there are few conditions less satisfactorily treated than this when it is well established.

Dickson concluded that probably in no traumatic condition is it as necessary to be constantly on the lookout for circulatory and nerve interference, as in injuries about the elbow in children, for failure to recognize the onset of ischemia means disaster for the patient and criticism for the medical attendant.

Nelson as recently as 1930 deplores the so few results of prophylactic surgical measures have been reported that it is hard to decide on a regime. Especially when it is all important to do so within the first twelve hours of the onset in order to be at all certain of a favorable outcome.

Girdlestone says "All the uncertainties which made it so formidable have, in recent years, been cleared away, and, now that we understand its causation, we can (almost always) prevent its development.

Thomas showed, by a study of the cases of ischemic contractures reported in the literature that this condition varies greatly in severity in different individuals and instances, and that in many of the cases we are dealing with complications of the primary trouble, secondary to involvement of some of the nerve trunks which produce disturbances which cannot be relieved by the treatment of the contracture only. So that the individual case must be carefully examined with the question of nerve involvement in mind, and this condition considered in determining the treatment, so that in one case a nearly perfect result is obtained and in another practically no improvement results.

Meyerding, is probably one of the best recent authorities on the subject of ischemic contracture, gives some very helpful and comprehensive prophylactic procedures and information.

He has concluded that when treatment involves acute flexion at the elbow, with or without circular bandaging, a cast or splint, careful observation every few hours for the first few days is demanded, after proper reduction of a fracture in this region, for pulse, cyanosis, temperature of the limb, and neurological signs .

In regards to the pulse it is well to remember that pulse loss may be insidious or precocious and it is particularly important to keep this in mind as was observed and further emphasized by Groves the importance of watching minutely for injury to and change in the soft tissues and treating them, particularly the circulation, and secondarily attending to the fracture.

Meyerding believes it is far better to take care of the soft tissues and eliminate intrinsic pressure before one tries to reduce the fracture with splints, casts or bandages and therefore add insult to injury, and this applies to manipulation and acute flexion as well.

In supracondylar fractures, which are treated by acute flexion, the extremity should be carefully and repeatedly examined during the first few hours for pulsation by comparing it with the opposite limb, also for color changes, swelling, and unrelieved pain. He warns also that especial care must be taken in cases in which splints or similar devices have been applied and in which the pain persists in spite of opiates. When swelling and cyanosis persist, and the circulation is obviously markedly impaired, drainage of the hematoma and

of the deep tissues may be of great benefit in preventing of the contracture.

Hot dressings and moderate extension may relieve the pressure and prevent the necessity of drainage, thus avoiding the compounding of a fracture. When severe symptoms persist, a deep incision and moist, warm dressings are indicated. When ischemic contracture has occurred and is diagnosed within a few hours of its occurrence, treatment of the fracture should by all means be abandoned and all attention be paid to improvement of the circulation. The above procedure in Meyerdings experience works very well. He further emphasizes that retention splinting should give the patient a sense of comfort and security.

In this connection Murphy gives some very sound and timely warning in regards to splinting and bandageing. He says, "Bandages and splints should never be applied for the purpose of reducing a fracture and rarely if ever for the maintainance of position by force after reduction. They are merely means of limiting voluntary, or mechanical disturbance after reduction is effected.

Murphy, Jones and others, believe the treatment of impending contracture by free incision of the deep fascia over the antecubital fossa where it occurs in the arm, is a well established procedure and Jepson in his experiments showed pretty conclusively that this applies else where in the body where this condition threatens to develop.

Kleinburg gives several practical suggestions in handling these cases. A limb which is deformed or diseased but not likely to swell we may apply with safety a plaster dressing over only a slight amount of protection, as a flannel bandage. In case of an injury, as a fracture, however, in which the trauma to the blood vessels and consequent swelling, it is essential to surround the affected part with some such material as sheet cotton. This material is very compressible and will allow for swelling without danger of undue compression. It is further advisable to leave the fingers exposed or at least accessible to observation.

We must instruct the patient that at all times the color of the fingers must be normal, and the patient should at all times be able to move them.

As regards the palsies the physician should make careful neurological examinations immediately and frequently. If the nerve is divided the results are always more satisfactory if the divided ends are sutured immediately or at least early, and this was particularly emphasized by Johnson.

When nerve function is being impaired by an encroaching cicatrix it should be freed as soon as possible it can be detected for best functional prognosis.

Eikenberry expressed the opinion that Volkmann's contractures are entirely too frequent, and the best way to prevent their occurrence is by being in very close touch with the patient, so that the splints can be readjusted at the first warning in the case of fractures. A few hours of neglect may lead to a condition that can never be repaired.

Dickson concluded that Volkmann's contracture offers almost insurmountable obstacles to cure, unless its onset is recognized early and measures taken at once to remove the cause.

When the contracture is of days or weeks duration, heat, massage and extension splints are of most value. active exercise in a warm whirl

pool bath ,followed by massage with gentle upward stroking, is indicated to relieve edema. Forceful stretching under anesthesia does more harm than good except in very early cases. Physical therapy and continuous stretching are of considerable benefit when carried out over long periods of time. Early splinting and continuous stretching are essential to prevent further deformity and disability. These procedures, in Meyerdings practice, have given the most satisfactory results.

It is the consensus of opinion of several writers since the condition was first described that the nerve is always involved and should be considered in the treatment of the deformity, even though it is secondary because it can ultimatly contribute to the deformity.

The following sage suggestion of Murphy so nicely summarizes this whole phase of the subject that I deemed it sufficiently interesting to repeat it. Murphy says, " Above all, however, your morning prayer when treating these cases should include, "Deliver me this day from excuting passive motion in a recent fracture of the elbo when that passive motion causes the patient severe pain," as

it increases callous, connective tissue formation,
fixation, adds trauma already present in the
superlative degree."

Medico-legal - It was Meyerding who very recently stressed the fact that Volkmann's contracture may result from injury when no treatment has been administered of any kind and this is highly significant from a medico-legal standpoint because the layman and some physicians believe it to be due to certain types of articles used in the treatment of fractures.

Groves in reviewing the case of Dr. Alcock vs Tyndall, suggested that the attending physician in treating a case of fracture be particularly careful to get as nearly perfect anatomical results as possible since it was demonstrated in the particular case in question that the public is particularly prone to take this as a sure sign of negligence on the part of the physician in attending the case? He also emphasizes the importance, from a medico legal standpoint, of remembering that there are only certain groups of muscles involved and therefore always some chance of partial restoration of function if properly treated.

Powers outlined the following points to keep in mind in this connection and I believe might be helpful since this condition is always

such a potential source of the possibility of malpractice suits.

- (a) Whether an ischemic contracture or paralysis is present. This should not be difficult on careful notation of the electrical behavior of the muscle involved.
- (b) Whether the tightness of the splints, if they are employed, was at fault or whether there was originally a lesion of the artery; this is determined by pressure or absence of pulse beat below the fracture.
- (c) Whether the attending physician did or did not give to the case the care that his calling requires.

It is pointed out by Jones and others that not all Volkmann's contractures are due to lack of care since they can occur despite all precautions.

Summary -

- (1) Volkmann's ischemic contracture is a very important orthopedic condition.
- (2) Condition not sufficiently emphasized.
- (3) Extremely disabling deformity, very typical.
- (4) Occurs most often in individuals under 20 years of age and involves the fore-arm in 90% of cases.
- (5) Etiology not well known. Process not reversible.
- (6) Impending contracture very amenable to treatment.
- (7) Can be produced by intrinsic or extrinsic factors.
- (8) Treatment very unsatisfactory after deformity is fully developed.
- (9) Can occur spontaneously, without fracture or therapeutic interference, particularly splinting.
- (10) Important condition from medico-legal standpoint.
- (11) Condition more frequent than reported.

Conclusion - Volkmann's ischemic contracture is an extremely interesting deformity first because it is such a distinct and uniform condition and secondly because it is so hopeless both to the patient and the physician.

It is a comparatively recently described and recognized condition, and for a long time the only interest it seemed to draw was its treatment and it was not investigated from an etiological factor very intensively until very recently and this I would suspect is due to the fact that it was and still is such a bug bear to the physician from a medico-legal stand point so that more effort has been made to establish its etiological factor.

The literature is not particularly voluminous but the accounts are very complete and surprisingly not very contradictory, in general with the possible exception of the etiology.

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